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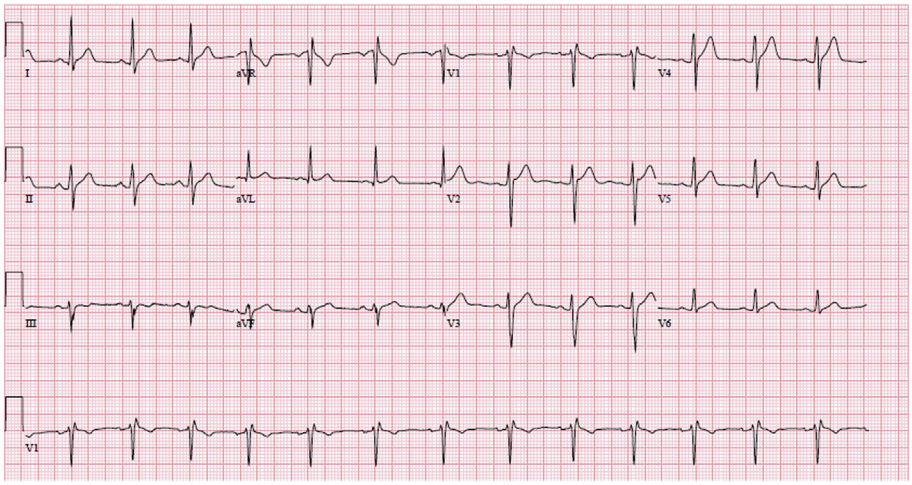

Figure 1. Normal sinus rhythm at 80 beats/min.

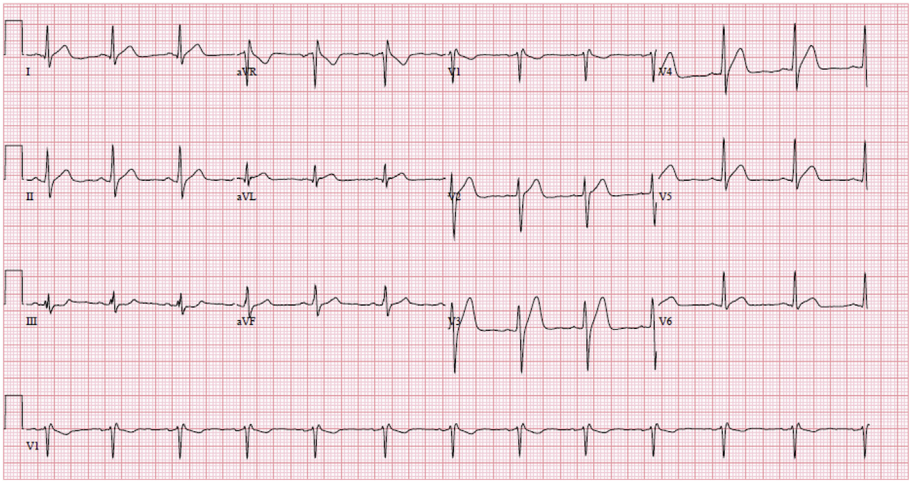

Figure 2. Normal sinus rhythm at 75 beats/min, with straightening of the concave ST segment anteriorly and newly enlarged T wave in V3 and possibly V4, not seen in Figure 1. Subtle ST segment depression in lead 3 is also appreciated.

[Ann Emerg Med. 2018;71:113-116.]

CASE PRESENTATION

A 27-year-old man with no reported medical history presented to the emergency department with an hour of crushing substernal chest pain radiating to his left arm, with associated nausea and diaphoresis. On examination, he was alert, oriented, and clutching his chest in significant distress. His blood pressure was 138/69 mm Hg, pulse rate was 75 beats/min, and respiratory rate was 18 breaths/min, with a normal temperature and oxygen saturation on room air. He endorsed tobacco use but adamantly denied any drug use, specifically, cocaine. A 12-lead ECG was obtained immediately (Figure 1) and blood was drawn for testing, including troponin levels. Because of the patient's concerning clinical presentation and unremitting pain, another 12-lead ECG (Figure 2) was obtained 10 minutes later.

Is the second ECG concerning for ischemia?

For the diagnosis and teaching points, see page 115.
To view the entire collection of ECG of the Month, visit www.annemergmed.com

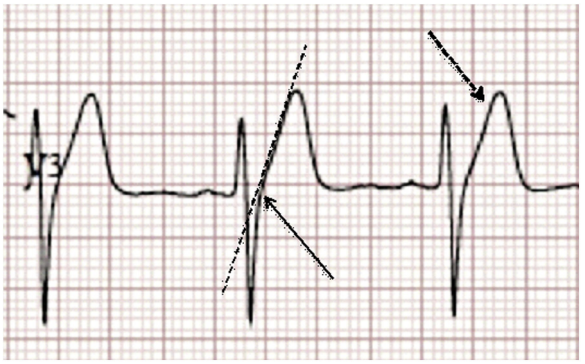

Figure 3. Lead V3 from the patient's second ECG (Figure 2) before cardiac arrest, highlighting the straightening of the ST segment (solid arrow and dotted line) and increase in amplitude of the T wave (dotted arrow).

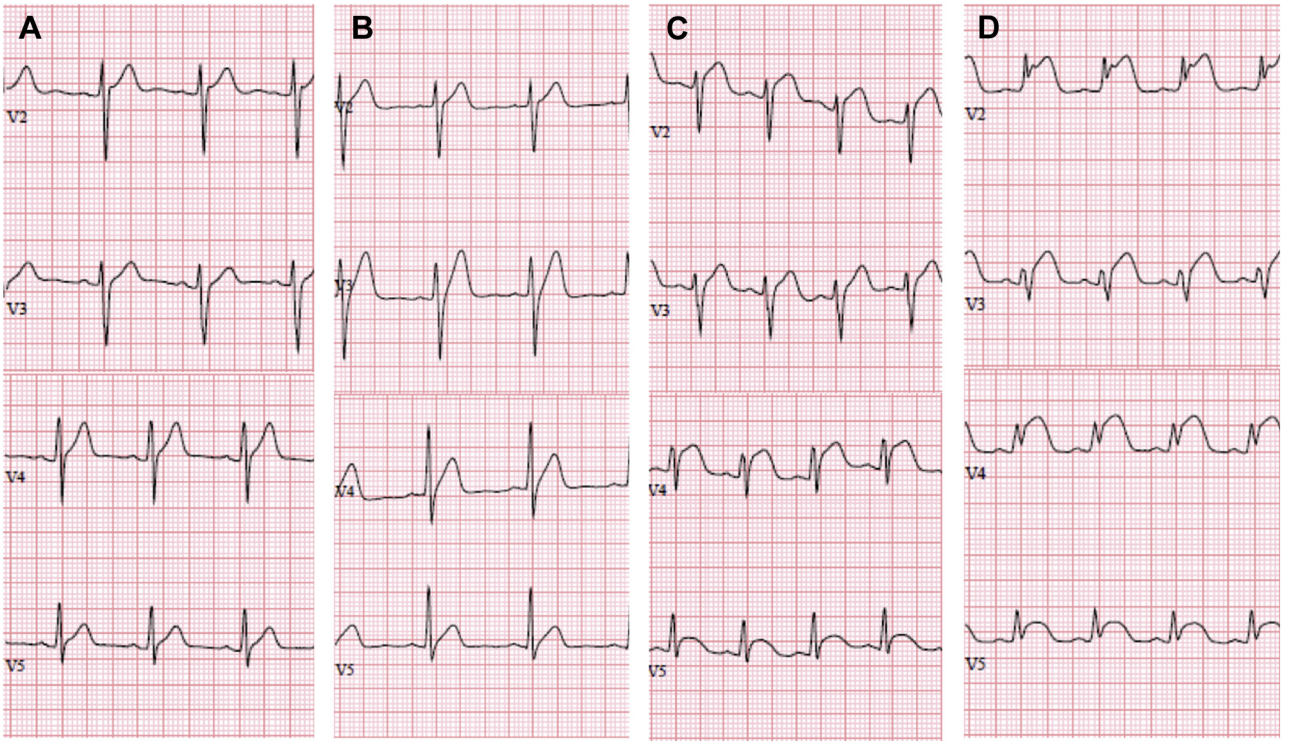

Figure 4. Leads V2 to 5 from the patient's 4 ECGs demonstrating STEMI evolution from (*A*) presentation and (*B*) second ECG before cardiac arrest, to (*C*) immediately after return of spontaneous circulation and (*D*) before patient's transport to the catheterization suite.

ECG OF THE MONTH
(continued from p. 113)

DIAGNOSIS

The first ECG (Figure 1) demonstrated normal sinus rhythm at approximately 80 beats/min, without ischemic changes. The second ECG (Figure 2) revealed normal sinus rhythm at approximately 75 beats/min, with straightening of the concave ST segments in the anterior leads and development of a newly enlarged T wave in lead V3 (Figure 3) and possibly V4. Subtle ST-segment depression was also noted in lead 3 (Figure 2).

CLINICAL COURSE

Shortly after the second ECG was obtained (Figure 2), a cardiology consultation was initiated. However, minutes later, the patient reported increased chest pain and suddenly became unresponsive, without a palpable pulse. The bedside monitor showed ventricular tachycardia. Cardiopulmonary resuscitation was immediately started, and the patient was defibrillated with 200 J within the first minute. Return of spontaneous circulation was achieved and the patient quickly became conscious and fully conversant. An ECG (Figure 4*C*) was obtained, showing new anterior and lateral ST-segment elevations. Antiplatelet and anticoagulant therapies were administered. A fourth ECG (Figure 4*D*) was obtained before transport to the catheterization laboratory, where an 80% to 90% proximal left anterior descending artery occlusion extending to the ostium was successfully stented. His initial troponin I level was less than 0.04 ng/mL and peaked at 43.46 ng/mL during hospitalization. Toxicologic screening was positive for cocaine.

DISCUSSION

In 2008, de Winter et al[1] described a new ECG sign consistent with proximal left anterior descending occlusion and evolution into anterior wall ST-segment elevation myocardial infarction (STEMI). The ST segment showed a 1- to 3-mm upsloping depression at the J point in the precordial leads that continued into tall positive symmetrical T waves.[1] Our patient's second ECG (Figure 2) did not fully demonstrate these changes. The ECG pattern described by de Winter et al[1] was different from hyperacute T waves, generally described as symmetric broad-based elevations of the T waves[2] that are transient and typically evolve into a classic STEMI pattern.[3] The ECG finding described by de Winter et al[1] was static and persisted from the initial recording at presentation until mechanical revascularization.[4]

Our case highlights several key points. Comparing the first 2 ECGs (Figures 1 and 2), we notice straightening of the ST segments anteriorly, with a sharp increase in amplitude of the T waves, especially in V3 (Figure 3). These changes differ from those described by de Winter et al[1] in that the ST segment was not depressed and the T wave was not completely symmetric; however, they both represented similar findings consistent with proximal left anterior descending occlusion. Despite lack of complete symmetry, our ECG findings represent changes more consistent with hyperacute T waves. Asymmetry of the T wave in early coronary occlusion might be due to early elevation of the ST segment, representing evolving transmural infarction,[2] as shown in Figure 4*C* and *D* with our patient. Comparing Figure 4*C* and *D*, we notice the ST-segment elevations in the anterior and lateral leads increased in convexity and amplitude. These findings are consistent with evolving STEMI.

Our case carries important implications for the emergency physician, emphasizing the need to be familiar with different ECG morphologies that may represent a STEMI or evolving infarction. Recognizing that evolving infarction may manifest as a spectrum of ECG changes, the straightening of the ST segment anteriorly in addition to development of tall T waves should raise concern for acute ischemia. This should prompt the emergency physician to confer early with cardiology for catheterization laboratory

activation and to start antiplatelet and anticoagulant therapies. Lack of this recognition can contribute to delays in care with percutaneous intervention or thrombolytic therapy, potentially leading to increased morbidity and mortality.

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REFERENCES

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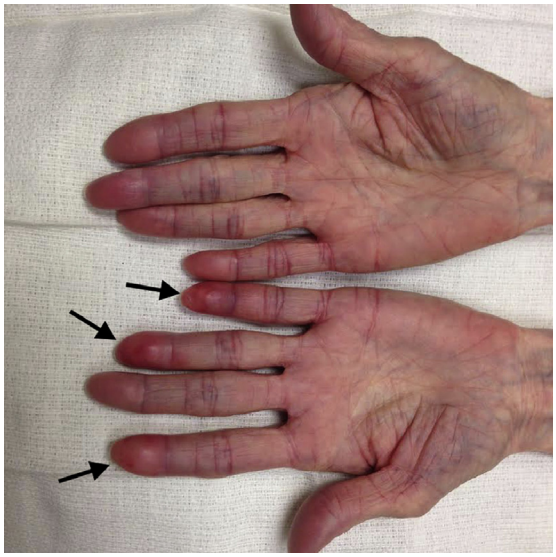

"Elderly Woman With Painful Swollen Fingers" by Bickel et al, March 2017, Volume 69, #3, pp. 297, 314.